REMARKS

Claims 1-21 were pending in the instant application. Claims 10-21 have been withdrawn from consideration as being directed to a non-elected invention. Claim 1 has been amended and claims 3 and 5 have been canceled. Thus, claims 1, 2, 4, and 6-21 will be pending upon entry of this amendment. Amendment and/or cancellation of the claims should not be construed as an acquiescence to any of the rejections.

Support for the amendments to the claims can be found throughout the specification and claims as originally filed. In particular, support for the amendment to claim 1 can be found at, for example, page 5, lines 23-27, page 16, lines 21-25, and page 17, lines 6-8 of the specification and in original claims 3 and 5. No new matter has been introduced.

Rejection of Claims 1, 2, 3 and 5-8 Under 35 U.S.C. §112, First Paragraph

Claims 1, 2, 3 and 5-8 have been rejected under 35 U.S.C. §112, first paragraph as allegedly failing to comply with the enablement requirement. In particular, the Examiner contends that, while the specification is enabling for an FcγRIIB promoter gene having polymorphic positions at residue -385 (C/C genotype) alone or in combination with residue -119 (T/A or A/A genotype), the specification does not enable "at least one polymorphic position within an FcγRIIB promoter gene" as is presently claimed.

Applicant respectfully traverses the foregoing rejection. Applicant respectfully submits that the present specification discloses more than one example of polymorphisms within the FcγRIIB promoter region and also discloses methods for identifying polymorphisms within the FcγRIIB promoter region (see, *e.g.*, page 13, line 21 through page 15, line 5 of the specification). In addition, the specification contains working examples which describe the identification of polymorphisms within the FcγRIIB promoter region as well as their association with systemic lupus

erythematosus (SLE) (see Examples 1-3). Based on the disclosure, Applicant respectfully submits that it would not require undue experimentation for one of ordinary skill in the art to identify additional polymorphisms within the FcγRIIB promoter region. However, in an effort to expedite prosecution, and in no way acquiescing to the Examiner's rejection, Applicant has amended claim 1 to specify that the reference polymorphic pattern comprises a C residue at position -385 and/or an A residue at position -119, thereby obviating the rejection. Accordingly, withdrawal of the rejection of claims 1, 2, 3 and 5-8 under 35 U.S.C. §112, first paragraph is respectfully requested.

Rejection of Claims 1, 2 and 8 Under 35 U.S.C. §112, Second Paragraph

The Examiner has rejected claims 1, 2 and 8 under 35 U.S.C. §112, second paragraph, as allegedly being indefinite for failing to particularly point out and distinctly claim the subject matter which Applicant regards as the invention. In particular, the Examiner contends that it is unclear what the criteria or parameters are for a comparison of a "test polymorphic pattern" and a "reference polymorphic pattern" in order to draw a conclusion regarding whether an individual is susceptible to development of SLE. The Examiner cites page 5, lines 23-27 of the specification, which states that "[i]f the test pattern matches the reference pattern, there is a statistically significant probability that the individual has or may develop SLE," and indicates that the claims do not recite that the "test pattern" matches the "reference pattern" as provided in the specification.

Applicant respectfully traverses the foregoing rejection and submits that claims 1, 2 and 8 are clear and definite. However, in an effort to expedite prosecution of the application, and in no way acquiescing to the Examiner's rejection, claim 1 has been amended. As amended, claim 1 is directed to a method for assessing whether an individual has or is susceptible to development of systemic lupus erythematosus comprising comparing a test polymorphic pattern with a reference polymorphic pattern and concluding whether the individual is susceptible to development of systemic lupus erythematosus wherein identity between at least one polymorphism included in the test polymorphic pattern and at least one polymorphism included in the reference polymorphic pattern indicates that the individual has or is susceptible to development of systemic lupus erythematosus. Applicant respectfully submits that claim 1 is clear and definite. Accordingly,

Applicant respectfully requests reconsideration and withdrawal of the foregoing rejection under 35 U.S.C. §112, second paragraph.

Rejection of claims 1, 2, and 8 Under 35 U.S.C. §103

The Examiner has rejected claims 1, 2, and 8 as allegedly being obvious in view of Kimberly et al. and Jiang et al. In particular, the Examiner contends that Kimberly et al. describes a method for determining whether the Fcγ (i.e., FcγRIIA) receptor allelic pattern of the patient corresponds most closely to known Fcγ receptor allelic patters of patients having autoimmune disease, such that one can determine if an individual has a predisposition for SLE. The Examiner admits that Kimberly et al. does not utilize at least one or at least two polymorphic positions within an FcγRIIB promoter gene as claimed in claims 2 and 8, respectively. The Examiner further contends that Jiang et al. describes the identification of polymorphisms within the FcγRIIB promoter gene, which can be utilized to determine the predisposition of an individual for SLE (*see* the Abstract; page 1686, lines 23-38; and Table 3). Therefore, the Examiner concludes that one of skill in the art would have combined the diagnostic method of Kimberly et al. with the polymorphisms identified in the FcγRIIB promoter taught in Jiang et al. to arrive at the present invention.

Applicant respectfully traverses the foregoing rejection. As set forth above, the pending claims are directed to a method for assessing whether an individual has or is susceptible to development of SLE comprising comparing a test polymorphic pattern comprising at least one polymorphic position within an FcγRIIB promoter gene of the individual, with a reference polymorphic pattern derived from a population of individuals having SLE, wherein the reference polymorphic pattern comprises a C residue at position -385 and/or an A residue at position -119; and concluding whether the individual is susceptible to development of SLE where identity between at least one polymorphism included in the test polymorphic pattern and at least one polymorphism included in the reference polymorphic pattern indicates that the individual has or is susceptible to development of SLE.

Kimberly et al. describes a diagnostic method for determining whether a patient has a predisposition to develop certain autoimmune diseases. The method described in Kimberly et al. generally includes identifying a characteristic allelic pattern or genotype for one or more Fcγ receptor genes and comparing the Fcγ receptor allelic pattern to the distribution of allelic patterns in test populations (see, for example, col. 4, lines 12-18 of the specification). In particular, Kimberly et al. describe diagnostic methods for determining predisposition to SLE which comprise identifying the presence of certain alleles of the FcγIIA gene in an individual (see, for example, claim 15 and col. 7, lines 14-25). Kimberly et al. do not teach or suggest methods for assessing whether an individual has or is susceptible to development of SLE using polymorphic patterns within the FcγRIIB gene. Moreover, Kimberly et al. do not teach or suggest methods for assessing whether an individual has or is susceptible to development of SLE wherein the reference polymorphic pattern comprises a C residue at position -385 and/or an A residue at position -119, as is claimed in the instant application.

Jiang et al. fails to make up for the deficiencies of Kimberly et al. Jiang et al. describes the finding that a murine FcγRIIB promoter allele containing certain nucleotide deletions in the NZB mouse strain is associated with abnormal down-regulation of FcγRIIB1 levels in germinal center B cells (see page 1689, left column and Fig. 3). Jiang et al. postulates that the deletion in the FcγRIIB promoter may possibly predispose to SLE through germinal center B cells abnormally down-regulating FcγRIIB1 expression (see Abstract). Jiang et al. fails to teach or suggest a method for assessing whether an individual has or is susceptible to development of SLE by comparing a test polymorphic pattern comprising at least one polymorphic position within an FcγRIIB promoter gene of the individual, with a reference polymorphic pattern derived from a population of individuals having SLE, wherein the reference polymorphic pattern comprises a C residue at position -385 and/or an A residue at position -119.

For a prior art reference (or references when combined) to render an application obvious, the prior art must teach or suggest all the claim limitations (M.P.E.P. 2143). In view of the foregoing, Kimberly et al. and Jiang et al., fail to teach or suggest the claimed methods.

Accordingly, Applicant respectfully requests reconsideration and withdrawal of the rejection of claims 1, 2, and 8 under 35 U.S.C. §103.

CONCLUSION

In view of the above amendments and remarks, it is respectfully requested that the application be reconsidered and that all pending claims be allowed and the case passed to issue. If there are any other issues remaining which the Examiner believes could be resolved through a Supplemental Response or an Examiner's Amendment, the Examiner is respectfully requested to contact the undersigned at the telephone number indicated below.

Respectfully submitted,

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